PROC (BAYL UNIV MED CENT)
2021;34(6):742–743

https://doi.org/10.1080/08998280.2021.1951072

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Scurvy in a patient with a restrictive diet

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ABSTRACT

Scurvy is a historically significant disease whose incidence has declined significantly with advancements in nutrition and access to varied foods. It is classically characterized by gingival bleeding, corkscrew hairs, and petechiae. In cases of severe deficiency, as seen in patients with years of a restricted diet, impaired connective tissue formation can lead to symptomatic, life-threatening bleeding diathesis. Risk factors for a restrictive diet in patients with unidentified bleeding diathesis should be recognized early to prevent significant morbidity.

KEYWORDS Ascorbate; ascorbic acid; bleeding diathesis; restrictive diet; scurvy; vitamin C

Unlike most animals, humans are unable to synthesize vitamin C (ascorbic acid) from glucose, and thus it is a dietary essential nutrient. Endogenously, vitamin C is notably used in collagen synthesis and as an antioxidant. While vitamin C deficiency, defined as serum levels <11 μmol/L, has an estimated prevalence of 8.4% of the US population,[1] true symptomatic scurvy is quite rare. Demographic characteristics of male gender and advanced age have been linked to vitamin C deficiency,[2] but it can also be seen in any patient with a restrictive diet. Patients deficient in vitamin C can present with fatigue, myalgias, hair and skin changes, and impaired wound healing. We describe a patient with a restrictive diet whose scurvy led to severe anemia and presented as bleeding diathesis that gradually improved with vitamin C supplementation.

CASE DESCRIPTION

A 33-year-old white man with a history of hypertension, morbid obesity (body mass index 50.24 kg/m^2), autism, acne vulgaris, and allergic rhinitis presented to the emergency department with complaints of fatigue, lightheadedness, and bilateral leg pain for 1 month. He had been taking amlodipine-benazepril, doxycycline, montelukast, and ibuprofen for his leg pain. He had no prior personal or family history of bleeding disorders. He denied any trauma. Due to his autism, he found any new foods intolerable and his diet consisted nearly exclusively of hamburgers, pasta, and cheese.

On admission, vital signs were normal. He had ecchymoses on his posterior thighs, as well as perifollicular hemorrhages and bleeding gingiva *(Figure 1)*. He had been seen 3 weeks earlier for similar complaints, where he had a normal hemoglobin and a computed tomography angiography of his lower extremities did not show any evidence of deep vein thromboses, vascular abnormalities, or hematoma. His hemoglobin was 5.1 g/dL, iron was 28 μg/dL, total iron binding capacity was 356 μg/dL, and ferritin was 75 ng/mL, consistent with iron deficiency anemia. His international normalized ratio was 1.2, and there was no evidence of intravascular hemolysis.

Despite repeated transfusions, the patient's hemoglobin failed to appropriately respond. Further workup revealed mild Factor VII activity deficiency, as well as a mixing study consistent with a weak inhibitor present. Additionally, upper endoscopy and colonoscopy disclosed linear stripes of gastric erythema with focal lamina propria hemorrhage.

The patient was started on supplemental oral vitamin C 500 mg daily. Vitamin C levels taken before treatment resulted at a level below quantifiable assay limits. No active bleed was discovered throughout the patient's admission. His hemoglobin stabilized after 12 units of packed red blood cells and 6 days of vitamin C supplementation. He was discharged home in stable condition. At outpatient follow-up, the patient reported improved symptoms. His hemoglobin continued to normalize with the successful addition of vegetable juice to his diet.

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The authors report no conflict of interest. Appropriate consent has been obtained for this case to be published.
Received May 30, 2021; Revised June 23, 2021; Accepted June 28, 2021.

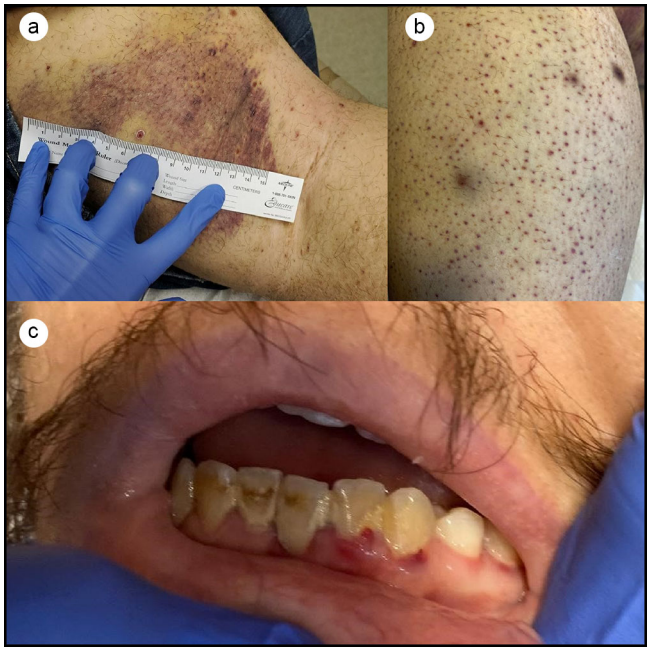

Figure 1. **(a)** Posterior right lower extremity ecchymosis. **(b)** Perifollicular hemorrhages on anterior right lower extremity. **(c)** Gingival bleeding.

DISCUSSION

Ascorbic acid is an essential nutrient required for collagen synthesis and repair. Though adequate levels of vitamin C can be obtained through consuming minimal fruits and vegetables, extremely restricted diets can lead to symptomatic scurvy within 2 months.[3] Diagnosis is largely clinical, with recognition of corkscrew hairs, perifollicular hemorrhages, impaired wound healing, and bleeding diathesis. Diagnosis may be aided with serum vitamin C levels, vitamin C tolerance tests, and follicular biopsy.[3] Once scurvy has been identified, treatment consists of 300 to 500 mg of vitamin C daily, and symptoms usually improve within 2 weeks.[4] Supplementation should be continued if the diet remains restricted. If left untreated, scurvy can lead to hemarthrosis, spontaneous bleeding, hemorrhagic pericarditis, arrhythmias, and potentially death.[3]

Collagen dysfunction in patients with scurvy can lead to damage of collagen-rich connective tissues including capillary beds, leading to microscopic extravasation of blood. In our morbidly obese patient, sitting was enough to cause trauma to the capillaries and lead to diffuse but clinically significant hemorrhage into his lower extremities. This microscopic bleed was likely present throughout the patient's body, as evidenced by his gastric biopsy showing hemorrhage into the mucosa. In addition to its role in collagen synthesis, vitamin C also promotes dietary nonheme iron absorption by reduction of ferric iron to its ferrous form that can be absorbed in the duodenum.[5] Though the absorption of heme iron is unaffected by vitamin C deficiency, this pathway may additionally contribute to iron deficiency anemia in scurvy.

Our case demonstrates that scurvy is a rare but important cause of bleeding diathesis, and a high index of suspicion should be held for patients with a restrictive diet, including those who are institutionalized, those with autism, and the elderly. Considering its safety profile, empiric treatment with vitamin C should not be delayed in patients with suspected deficiency.

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